Silicosis—An Over View

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Abstract—Silicosis is an occupational health disease affecting lungs caused by inhalation of silica dust. It leads to lung tumors, pulmonary tuberculosis and various other serious health hazards such as mycobacterial infection, fungal infection, certain autoimmune diseases. It is mostly found in workers exposed to silica such as mining, sandblasting, quarry, ceramics as well as grinders, stone cutters, brick workers, workers in the oil and gas industry, pottery workers, fiberglass manufacturing. The disease can be diagnosed by chest X-ray examination. Industrialization has lead to rapid spread of the disease. The article describes types of silicosis, diagnosis, pathophysiology, prevention, treatment, and regulatory measures by the government bodies. It is a preventable disease. The disease needs to be further investigated towards prevention, treatment and diagnosis.

Keywords—Silicosis, lung disease, prevention, regulations.

INTRODUCTION

Silicosis (also known as miner’s phthisis, grinders asthma, potter’s rot and other occupation-related names [1], or by the invented name pneumo silicosis) is a form of occupational lung disease caused by inhalation of crystalline silica dust, and is marked by inflammation and scarring in the form of nodular lesions in the upper lobes of the lungs [2; 3]. It is a type of pneumoconiosis. The symptoms of acute silicosis include cough, shortness of breath, cyanosis, and fever. It is often misdiagnosed as fluid in lungs or pneumonia or infectious tuberculosis.

Silicosis resulted in 46,000 deaths globally in 2013 down from 55,000 deaths in 1990 [4; 5]. Prosector Achille Visconti (1836–1911), in the Ospedale Maggiore of Milan [6] coined the name silicosis which is originated from Latin word silex, or flint in 1870. The ancient Greeks and Romans [7], recognized respiratory problems caused by inhaling dust. Lung problems in miners caused due to dust was noted in a mid-16th century written book by Agricola. The rapid industrialization lead to increased production of dust as people left hand tools and shifted to machines. In 1973, Mazzini noted asthmatic symptoms and sand-like substances in the lungs of stone cutters. The factors that contributed initially to the rapid spread of silicosis were introduction of, pneumatic hammer mill in 1897 and sand blasting in 1904 [8]. Silicosis resulted in 46,000 deaths in 2013 down from 55,000 deaths in 1990 [6].

EPIDEMIOLOGY

Occupational silicosis

Silicosis is the most common occupational lung disease worldwide; it occurs everywhere, but is especially common in developing countries [9]. From 1991 to 1995, China reported more than 24,000 deaths due to silicosis each year [1]. In the United States, it is estimated that between one and two million [10] workers have had occupational exposure to crystalline silica dust and 59,000 of these workers will develop silicosis sometime in the course of their lives [1]. Silicosis is very rarely seen in US according to CDC data [10]. The incidence of deaths due to silicosis declined by 84% between 1968 and 1999, and only 187 deaths in 1999 had silicosis as the underlying or contributing cause [10]. Additionally, cases of silicosis in Michigan, New Jersey, and Ohio are highly correlated to industry and occupation [11]. In earlier days even silicosis existed but the rapid industrialization with the use of pneumatic drilling and mine explosions leading to raise in silica dust.

In the United States, a 1930 epidemic of silicosis due to the construction of the Hawk's Nest Tunnel near Gauley Bridge, West Virginia caused the death of at least 400 workers. Other accounts place the mortality figure at well over 1000 workers, primarily African American transient workers from the southern United States [12]. Making an exact mortality account was hard as some of the workers left the region and workers suffering from the disease were fired. America’s worst industrial disaster was “The Hawks Nest Tunnel Disaster” [12]. Some men started growing mustache caleed miner’s mustache to protect from extra silica.

Chronic simple silicosis has been reported to occur from environmental exposures to silica in regions with high silica soil content and frequent dust storms [13]. Nevada was ruined by a dry mining establishment, that caused hundreds of deaths. It was nick named The widow maker (Delamar Ghost town) after the incident.

The problem was tried to overcome by the attachment of a water spraying nozzle to the drill. But this caused conversion of dust to mud inhibiting mining work. The workers from various industries such as quarry, ceramics, sandblasting, foundry, grinders, stone cutters, stone countertops, brickworkers, tombstone workers, pottery, fiber glass, flint knappers, oil and gas industry are generally affected by silicosis proving it an occupational hazard. Brief or casual exposure to low levels of crystalline silica dust are said to not produce
silicon dioxide \(\text{SiO}_2\)

- **Crystalline silica** exists in 7 different forms (polymorphs), depending upon the temperature of formation. The main 3 polymorphs are quartz, cristobalite, and tridymite. Quartz is the second most common mineral in the world (next to feldspar) [9].
- **Microcrystalline silica** consists of minute quartz crystals bonded together with amorphous silica. Examples include flint and chert.

Crystalline silica is more toxic than amorphous silica. It is not inert biologically. Diatomite on heating gets converted to cristobalite or tridymite.

- **Skeletons of diatoms and vitreous silica on heating and rapid cooling produce amorphous silica which contains diatomite or kieselguhr. Amorphous silica can also be obtained from the heating and rapid cooling of crystalline silica.**
- **Silica flour** is nearly pure \(\text{SiO}_2\) finely ground. The various ways in which silica flour can be used include filler for cosmetics, abrasive, paint extender, buffer and polisher. Silicosis may be chronic or acute caused by inhalation of fine silica dust measuring <10μ in diameter. The fine dust contains crystalline silica in forms tridymite, cristobalite or alpha-quartz [9].

### TYPES OF SILICOSIS

Silicosis can be categorized into various classes depending on its onset, progress and severity of disease [5].

- **Chronic simple silicosis**
  The most common type of silicosis, which results from long-term exposure (10 years or more) to relatively low concentrations of silica dust. It appears 10–30 years after first exposure to silica. Patients with this type of silicosis, in the early stages do not have distinguishable signs or symptoms of disease, but abnormalities in lungs can be detected by X-ray examination. The common findings include chronic cough and exertional dyspnoea (shortness of breath). Radiographical examination reveals profusion of small (<10 mm in diameter) rounded opacities, typically present predominantly in the upper lung zones.
- **Accelerated silicosis**
  This form of Silicosis develops 5–10 years after first exposure to higher concentrations of silica. It’s symptoms and x-ray findings are like chronic simple silicosis. The disease progresses more rapidly. Patients are at greater risk for disease complication with progressive massive fibrosis (PMF).
- **Complicated silicosis**
  Silicosis becomes "complicated" by gradual severe scarring (glomerate silicosis or progressive massive fibrosis), where the small nodules become confluent, reaching a size of 1 cm or greater. PMF is associated with respiratory impairment and more severe symptoms. It can be further complicated by other lung disease, such as tuberculosis, non-tuberculous mycobacterial infection, and fungal infection, certain autoimmune diseases, and lung cancer. Complicated silicosis is more common with accelerated silicosis than with the chronic variety.
- **Acute silicosis**
  It develops due to exposure to high concentrations of respirable silica dust in a few weeks to 5 years, also known as silicoproteinosis. The symptoms include rapid onset of severe shortness of breath, cough, weakness, and weight loss, often leading to death. The x-ray examination reveals a diffuse alveolar filling with air bronchograms, described as a ground-glass appearance. They resemble pneumonia, alveolar hemorrhage, pulmonary edema, and alveolar cell cancer in lungs [7].

### PATHOPHYSIOLOGY

Small silica dust particles when inhaled, embed deeply into the tiny alveolar sacs and ducts in the lungs, where oxygen and carbon dioxide gaseous exchange takes place. The lungs cannot clear the dust out by mucous or coughing. On accumulation of silica dust in the lungs, macrophages that ingest the dust particles create an inflammation response by releasing tumor necrosis factors, leukotriene B4, interleukin-1, and other cytokines. Further, stimulate fibroblasts to proliferate and produce collagen around the silica particle, thus
resulting in fibrosis leading to the formation of the nodular lesions. The NALP3 inflammasome mediates the inflammatory effects of crystalline silica. Nodular silicosis contains fibrotic nodules which can be seen under polarized light with concentric "onion-skinned" arrangement of collagen fibers, a cellular peripheral zone, with lightly birefringent particles, and central hyalinization. The lung tissues react specifically to silica and form nodules [8]. In acute silicosis, microscopic pathology shows a cellular infiltrate of the alveolar walls and a periodic acid-Schiff positive alveolar exudate (alveolar lipoproteinosis) [4].

SIGNS AND SYMPTOMS

Chronic silicosis is slow to develop, signs and symptoms appear years after exposure [1].

- Cough, often persistent and sometimes severe
- Dyspnea (shortness of breath) exacerbated by exertion
- Tachypnea (rapid breathing) which is often labored
- Fatigue
- Loss of appetite and weight loss
- Fever
- Chest pain
- Gradual deep shal lows in nails eventually leading to cracks as protein fibers within nail beds are destroyed.
- Gradual darkening of skin (blue skin)

In advanced cases, the following may also occur
- Respiratory insufficiency
- Cor pulmonale (Right ventricle heart disease)

Patients with silicosis are susceptible to tuberculosis (TB) known as silicotuberculosis. The reason for the 3 fold increased risk is not well understood. It is assumed that pulmonary macrophages are damaged by silica, inhibiting their ability to kill mycobacteria. Industrial workers with prolonged silica exposure, but without silicosis, are at an increased risk for TB [2]. Pulmonary complications of silicosis include chronic bronchitis, non-tuberculous mycobacterium infection, fungal lung infection, airflow limitation (indistinguishable from that caused by smoking), pneumothorax, and compensatory emphysema. Especially in acute or accelerated silicosis, there is a close association between silicosis and certain autoimmune diseases, including scleroderma, nephritis, and Systemic Lupus Erythematosus.

The International Agency for Research on Cancer (IARC), in 1996, classified crystalline silica as "carcinogenic to humans" after reviewing the medical data, best seen in cases with silicosis, with relative risks for lung cancer. Numerous studies have published confirming this risk [2]."

DIAGNOSIS OF SILICOSIS

Silicosis can be diagnosed by three elements. First, the patient exposure to sufficient silica dust to cause this illness. Second, chest X-ray that reveals silicosis. Third, there are no underlying illnesses causing the abnormalities. Physical examination is unremarkable in complicated disease. The examination findings are not specific for silicosis. Pulmonary function testing may reveal airflow limitation, reduced diffusion capacity, restrictive defects, mixed defects, or may be normal (Especially without complicated disease). Tissue biopsy is not required in most cases of silicosis diagnosis, but may be necessary in some cases, to exclude other conditions. Chest X-ray (Fig.1) will confirm the presence of small (< 10 mm) nodules in the lungs, especially in the upper lung zones for uncomplicated silicosis. ILO classification states profusion 1/0 or more than that with shape p,q, and r indicates damaged lungs due to silicosis.

Profusion and lung zone involvement increases with disease progression. Particularly in the upper lung zones, in advanced cases of silicosis, large opacity (> 1 cm) occurs from coalescence of small opacities. There is a compensatory emphysema with retraction of the lung tissue. Enlargement of the hilum occurs commonly in chronic and accelerated silicosis. The nodes will calcify circumferentially, producing so-called "eggshell" calcification in about 5–10% of cases. The finding is not diagnostic (Pathognomonic) of silicosis. In some cases, the pulmonary nodules become calcified. A computed tomography or CT scan can also provide detailed analysis of the lungs, and reveals cavitation because of concomitant mycobacterial infection (Fig. 1) [21].

PREVENTION

The best way to prevent silicosis is to identify activities that produce respirable crystalline silica dust and eliminate or control it ("Primary prevention") in work place. Water spray, dry air filtering techniques are often used where dust comes out [22]. Jaggery (A traditional sugar) was found to have preventive action in Lucknow, India against silicosis in experiments conducted on rats [8].

TREATMENT

Silicosis has no cure at present and is a permanent disease [4]. Treatment options include alleviating the symptoms and preventing progression of the condition further.

- Stop exposure to silica dust, airborne silica, and other lung irritants, including tobacco smoking further.
- To treat bacterial lung infection antibiotics should be used.
- Use cough suppressants.
- For persons with positive tuberculin skin test or IGRA blood test, Tuberculosis (TB) prophylaxis should be undergone.
- Prolonged multi-drug regimen anti-tubercular drugs for those with active TB.
- Chest physiotherapy in patients to help the bronchial drainage of mucus.

Fig. 1: Chest X-ray showing uncomplicated silicosis
In patients with hypoxemia, Oxygen administration
To facilitate breathing use of bronchodilators.
The most effective treatment is lung transplantation, to replace the damaged lung tissue but is risky from consequences of long-term immunosuppression (e.g., Opportunistic infections).
To treat acute silicosis, broncho alveolar lavage should be carried out to alleviate symptoms, but does not decrease overall mortality [4].

Experimental treatments include:
- Inhalation of d-penicillamine, powdered aluminium, and polyvinyl pyridine-N-oxide.
- Corticosteroidal therapy.
- Administration of Chinese Herbal Kombucha
- The use of herbal extract tetrandrine, but may slow progression of silicosis [23].

REGULATIONS
In order to prevent silicosis, lung cancer, and other silica-related diseases in March 2016 OSHA officially mandated that companies must provide certain safety measures for employees who work with silica [20].

Key Provisions
- Permissible exposure limit (PEL) for respirable crystalline silica should be reduced 50 micrograms per cubic meter of air, averaged over an 8-hour shift.
- Engineering controls such as ventilation or water to limit worker exposure to the PEL should be used; when engineering controls cannot adequately limit exposure respirators should be provided; worker access to high exposure areas should be limited; develop a written exposure control plan, offer medical exams to highly exposed workers, and train workers on silica risks and how to limit exposures.
- Information about lung health should be given to workers who are very much exposed to silica.
- Provide flexibility to help employers — especially small businesses — protect workers from silica exposure [20].

Compliance Schedule
Both standards contained in the final rule took effect on June 23, 2016, after which industries have one to five years to comply with most requirements, based on the following schedule:
- If the construction dated, June 23, 2017, one year after the effective date.
- In case of general Industry and maritime dated June 23, 2018, two years after the effective date.
- In case of hydraulic fracturing dated June 23, 2018, two years after the effective date for all provisions except Engineering Controls, which have a compliance date of June 23, 2021 [20].

CONCLUSION
Silicosis threatens millions of people around the world and remains a crucial public health problem. The incurable, painful chronic disease, is spreading rapidly in emerging economies around the globe from old industrial regions. Primary prevention is the optimum form of prevention. Silicosis and silica-related diseases are preventable. Every country and region should work on preventive measures to meet the goal set by WHO and OSHA to eliminate silicosis by 2030.

REFERENCES
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